of S100 and NSE and had suffered an intracerebral hemorrhage;

Fig. 7 illustrates that patient SJ-2 had elevated MBP, Tm and S100 upon presentation and that the MBP and S100 levels continued to increase with time indicating a cerebral infarct with the stroke increasing over time;

Fig. 8 illustrates that patient SJ-18 presented with a TIA which evolved into a stroke. Tm was in the normal range indicating that the cerebral vasculature was not compromised and thus indicating that the patient was a good candidate for thrombolysis;

Fig. 9 illustrates that patient SM-8 presented with a cerebral infarct and, with Tm in the normal range, was a good candidate for thrombolysis since the endothelial vasculature was not compromised;

B2 cont

Fig. 10 illustrates that patient SJ-1 had a cerebral infarct and because of the elevated Tm level was at risk of hemorrhage if given thrombolytics because of the endothelial vasculature being compromised.

IN THE CLAIMS:

Claim 21 (Amended). A method for the differential diagnosis of ischemic and hemorrhagic cerebral events comprising:

B3

a. analyzing a body fluid of a patient to detect presence and concentration level of one or more ischemic marker proteins selected from the group consisting of myelin basic protein (MBP), the beta isoform of S100 protein (S100), neuronal specific enolase

(NSE) and combinations thereof,

b. analyzing a body fluid of said patient to detect presence and concentration level of a brain endothelial cell membrane protein, and

c. comparing the concentration level of any proteins detected in steps (a) and (b) to specific threshold values to verify the presence of statistically significant concentrations thereof of at least about two standard deviations above normal levels; and

d. assessing patient condition by comparing said presence or absence of statistically significant concentrations of said proteins in accordance with an analytical flow chart;

whereby differential diagnosis of an ischemic or hemorrhagic cerebral event is enabled.) scope any one effective measure of ischemic — inablement if one is correlated.

23 (Amended). A method as defined in claim 21 wherein said body fluid is selected from the group consisting of blood, blood [products] components and cerebrospinal fluid.

24 (Amended). A method as defined in claim 21 wherein said brain endothelial cell membrane protein is selected from one or more of the group consisting of Thrombomodulin, Glucose Transporter I in the dimeric or tetrameric form, Neurothelin, Gamma Glutamyl Transpeptidase, P-glycoprotein and combinations thereof.

26 (Amended). A method as defined in claim 21 further including:

analyzing said body fluid to detect presence and concentration level of a secondary marker protein which is cell type specific with respect to one of said myelin basic protein, beta isoform of S100 protein or neuronal specific enolase whereby the time of onset of a hemorrhagic or ischemic cerebral event can be determined.

27.(Amended). A method as defined in claim 26 wherein said secondary marker protein has a higher molecular weight than said corresponding myelin basic protein, beta isoform of S100 protein or neuronal specific enolase.

28 (Amended). A method as defined in claim 21 wherein each of said analyses is carried out on a single sample of body fluid.

Claim 34 (New). The method in accordance with claim 21 wherein said step of assessing patient condition includes:

1) initially concluding that a brain injury has occurred when one or more proteins are present;

2) further concluding that said brain injury is a TIA if only NSE is present;

3) further concluding that said brain injury is a lacunar infarct if only a brain endothelial cell membrane protein is

present;

4) further concluding that said brain injury is an intracerebral hemorrhage if MBP is present at a level equal to or greater than about 200 times normal levels;

5) further concluding that said brain injury is a cerebral infarct if S100 is present; and

6) further concluding that said brain injury is a subarachnoid hemorrhage if S100 and NSE are present.

Claim 35 (New). The method in accordance with claim 21 wherein said step of assessing patient condition includes:

determining when a brain endothelial cell membrane protein is present in addition to at least one ischemic marker protein and thereby concluding that an evolving cerebral infarct has occurred wherein the patient is a poor candidate for thrombolysis.

Claim 36 (New). The method in accordance with claim 21 wherein said step of assessing patient condition includes:

determining when S100 is present in addition to an increasing concentration of NSE absent the presence of a brain endothelial membrane cell protein and thereby concluding that an evolving cerebral infarct has occurred wherein the patient is a good candidate for thrombolysis.

Claim 37 (New). The method in accordance with claim 21 wherein said step of assessing patient condition includes:

determining when S100 is present alone or in addition to an increasing concentration of NSE or a brain endothelial membrane cell protein and thereby concluding that an evolving cerebral infarct has occurred wherein the patient is a poor candidate for thrombolysis.

Claim 38 (New). The method in accordance with claim 21 wherein said step of assessing patient condition includes:

determining when S100 is present in addition MBP at a level greater than two standard deviations and thereby concluding that severe cerebral edema is present.

Claim 39 (New). The method in accordance with claim 21 wherein said step of assessing patient condition includes:

determining when NSE is present along with at least one additional protein and thereby concluding that an evolving cerebral infarct has occurred; and

further determining if an elevated level of a brain endothelial cell membrane protein is present wherein the patient is a poor candidate for thrombolysis.